Neutrophil Elastase-mediated proteolysis activates the anti-inflammatory cytokine IL-

36 Receptor antagonist.

Tom Macleod¹, Rosella Doble¹, Dennis McGonagle^{2,3}, Christopher W. Wasson¹,

Adewonuola Alase², Martin Stacey¹, Miriam Wittmann*^{2,3,4,}

¹School of Molecular and Cellular Biology, Faculty of Biological Sciences, University of

Leeds, Leeds, UK

²Leeds Institute of Rheumatic and Musculoskeletal Medicine (LIRMM), University of Leeds,

UK

³National Institute of Health Research (NIHR) LMBRU, Chapel Allerton Hospital, Leeds

⁴Centre for Skin Sciences, Faculty of Life Sciences, University of Bradford, UK

*Corresponding Author:

Miriam Wittmann, MD

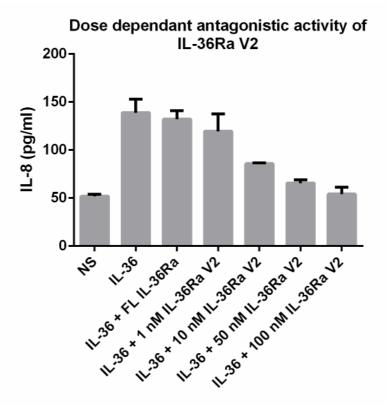
Chapel Allerton Hospital, LMBRU

Chapeltown Road, Leeds

LS7 4SA, UK

Email: M.Wittmann@leeds.ac.uk

Phone: ++44 113 392 4483



Supplementary Figure S1: Antagonistic activity of IL-36Ra V2 is dose dependant

Keratinocytes were stimulated with varying concentrations of IL-36Ra V2 in the presence of active agonist (10 nM). After 48 hours of treatment IL-8 concentrations analysed by ELISA. Mean +/- SEM is depicted. NS = non-stimulated, FL = full length. n=2.